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IN THIS ISSUE

Dr. McReynolds of Houston Elected President of Texas Orthopaedic Association ..... Page 335

New Mexico General Practitioners to Hold Summer Clinic in Ruidoso ..... Page 339

Santa Fe Seminar ..... Page 340

Porphyrin and Magnesium Supplementation in Collagen and Degenerative Disease ..... Page 348

The President's Page "I Am A Doctor" ..... Page 353

Clinical Pathological Conference, R. E. Thomason General Hospital, El Paso ..... Page 355

COMPLETE CONTENTS ON PAGE 330

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Contents

Dr. McReynolds of Houston Elected President
of Texas Orthopaedic Association ..... Page 335

New Mexico General Practitioners to hold
Summer Clinic in Ruidoso ..... Page 339

Santa Fe Seminar—Eclampsia and Preeclampsia,
St. Vincent's Hospital, Santa Fe, N. M. ..... Page 340
Harry D. Ellis, M.D., Chairman
Case Summaries by Fred Soldow, M.D.
Seminar Summary by Raymond L. Young, M.D.

Porphyrin and Magnesium Supplementation in
Collagen and Degenerative Disease ..... Page 348
By R. P. Watterson, M.D., Scottsdale, Arizona

The President's Page—"I Am A Doctor" ..... Page 353
By Russell L. Deter, M.D., El Paso

Clinical Pathological Conference,
R. E. Thomason General Hospital, El Paso ..... Page 355
F. P. Bornstein, M.D., Editor
Presentation of case by Victor Blanco, M.D.

New Concept of Retirement Inaugurated
in Brownsville Home ..... Page 361

Coming Meetings

The Society of Nuclear Medicine, Seventh Annual Meeting, Stanley Hotel, Estes Park, Colorado, June 22-25, 1960.

Postgraduate Course, Dermatology for General Practitioners, University of Colorado Medical Center, Denver, July 14-16, 1960.

Postgraduate Medical Assembly of South Texas, Shamrock Hilton, Houston, July 18-20, 1960.

New Mexico Chapter, American Academy of General Practitioners, Summer Clinic, Ruidoso, N. M., July 18-20, 1960.

Postgraduate Course in Pediatrics, University of Colorado School of Medicine, The Stanley Hotel, Estes Park, Colorado, Sept. 1-6, 1960.

Southwestern Medical Association, 42nd Annual Meeting, Hotel Hilton, El Paso, Oct. 20-22, 1960.

Southwest Obstetrical and Gynecological Society, Annual Meeting, Riviera Hotel, Las Vegas, Nev., Nov. 7-8, 1960.

Texas Orthopaedic Association, Galveston, Texas, April 24, 1961.

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SOUTHWESTERN MEDICINE

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MEETINGS

Dr. McReynolds of Houston Elected President of Texas Orthopaedic Association

Dr. I. S. McReynolds of Houston was elected president of the Texas Orthopaedic Association at its annual meeting in Fort Worth April 11, 1960, in connection with the annual convention of the Texas Medical Association. He is associate professor of orthopaedic surgery at Baylor University College of Medicine and clinical associate professor of orthopaedic surgery at the University of Texas Postgraduate School of Medicine.

Other new officers are Dr. Herbert Hipps, Waco, vice-president; and Dr. Margaret Watkins, Dallas, secretary-treasurer. The retiring president was Dr. David M. Cameron of El Paso.

Guest speakers at the annual orthopaedic meeting were Dr. Don O'Donoghue of Oklahoma City and Dr. W. H. Bickel of Rochester, Minnesota.

Resolutions Approved

Resolutions were approved in memoriam of Dr. William E. Barker, orthopaedic surgeon from Longview, who died February 5, 1960, at the age of 54; and in memoriam of Dr. Duncan C. McKeever, 53, Houston orthopaedic surgeon, who was fatally injured in an automobile accident on October 13, 1959.

The association now has 146 members and has the third largest membership of orthopaedic surgeons of any state in the nation.

The 1961 meeting is scheduled for April 24 in Galveston.

Dr. McReynolds was born in Pikeville, Tenn., and was graduated from the University of Tennessee College of Medicine in 1931. He took his internship at the U. S. Naval Hospital in New York in 1931-1932, and served for two years as junior medical officer in the Medical Corps of the U. S. Navy.

Serves Fellowship

He then served a fellowship in orthopaedic surgery at Carrell-Driver-Girard Clinic and Scottish Rite Hospital for Crippled Children, Dallas, in 1934 and 1935; and served as ortho-

Dr. McReynolds

chester Royal Infirmary in Manchester, England, from 1935 to 1936. He also served a fellowship paedic assistant to Sir Harry Platt at the Man- in orthopaedic surgery at the Willis C. Campbell Clinic, Memphis, Tenn., in 1936-1937; and spent three months in the Surgical-Pathological Department of Johns Hopkins Hospital, Baltimore, in 1937.

Dr. McReynolds entered the private practice of orthopaedic surgery in Houston in 1938. During World War II he served as a Lt. Colonel in the U. S. Army Medical Corps from 1943 to 1946.

Dr. McReynolds is a fellow of the American College of Surgeons, a past president of the Clinical Orthopaedic Society, and a member of the Houston Surgical Society, Houston Orthopaedic Society, Southern Medical Association, Southwestern Surgical Congress, Industrial Medical Association, International Society of Orthopaedic Surgery, and Traumatology (SICOT).

He and his wife have three sons; John, who is a senior at Princeton University; Bruce, age 15; and Angus, age nine. Their two daughters are Katherine, who is a freshman at Vassar College, and Heather, age six. He and his family reside at 301 Sage Road in Houston.

Dr. Cameron's Talk

The retiring president, Dr. Cameron, spoke on "Orthopaedic Surgery—an Art". His address follows:

From "Time whereof the memory of man runneth to the contrary," doctors have been admonished: "be learned in the Art and Science of your profession, and practice them." Daily the science bespeaks itself through multitudinous facts, neatly catalogued, for good or evil, requiring none but the abilities of reading and understanding to make them a part of our scientific learning. Webster defines Science shortly as "systematized knowledge of facts of the world which show the operation of general laws." But what is the Art? That which Hippocrates, 25 hundred years ago pledged to teach "by precept, oral teaching and every other mode of instruction." Again a Websterian definition—"Skill in performance, acquired by experience, study or observation"—This is the art in its most abbreviated form.

Philosopher's Approach

The philosopher's approach to the art helps explain this difficult to define attribute. He considers it to be a conscious activity which does three things: 1st—It manipulates or arranges natural materials, 2nd—It has purpose, and 3rd—The result thereof is regarded as an end which justifies its own existence. Here "Art" has traditionally been contrasted with "Nature." For example, as beautiful as a sunset may be it is not considered art; it is a natural phenomenon.

However, the skilled reproduction of it on canvas is art, fulfilling the philosophical criteria of 1) arrangement of natural material, 2) purposive and finally 3) the results justify its own existence. The philosopher further interprets the art as an intensified expression of experiences (or science). Here the doctor contributes his personality, knowledge, memory and imagination to mold environment, adds to it, and supplies the stuff of which the culture of a people is made.

Welfare and Happiness

Furthermore, there is a general enhancement of welfare and happiness with objective realization and subjective reaction. Thus are the learned definitions of the Art. Those who have practiced medicine, and love it, know there must be a warmer description. And indeed there is; words alone are inadequate, but they must be used.—personality, humility, reverence, honor, responsibility, curiosity, maturity. Of personality. Without patients the most brilliant scientist will go for naught. Annually training centers belch forth thousands of brilliant scientists into the medical world. Until they can become personable enough to be known, to be convincing to be human, or in other words sell themselves and their knowledge, they are not doctors. These are the traits of the medicine man with his medicine show of yesteryear. Indeed an art, however ours is based on a firm foundation of scientific fact and learning.

Traditional Dignity

Few will be medical politicians but all of us daily represent our profession to the public. Our conduct with traditional dignity, honor, humility and responsibility does more to retain our enviable place of respect among men and it is more forceful than any laws that legislators can impose. With Humility comes Reverence. Never was a doctor more confident of his knowledge than the day of his graduation. With passage of time and putting facts to the test, his learning becomes related to experience. Under certain circumstances two plus two produces five, as well as four. And the realization there is a power "greater than thou", referred to variously as "Mother Nature", "natural forces," or God. With humility comes wisdom. The wise doctor knows he learns but the rules of healing, abides by them, and this force "greater than thou" does the healing. He

ORTHOPAEDIC SPEAKER — Dr. Don O'Donoghue of Oklahoma City, left, who spoke at the annual session of the Texas Orthopaedic Association at Fort Worth April 11, 1960, is shown in a relaxing moment with Dr. C. F. Gregory of Dallas.

who learns this fact early has many years of service since times does not come free.

Likewise honor and responsibility are related one unto the other. Impetuous youth rapidly develops a sense of obligation as to the conduct befitting a respected professional man and becomes accountable for things as well as patients under his management and care; furthermore discharging community responsibilities and honorably understanding the value of and respect for human dignity which is the common denominator of us all.

Curiosity implies not only the desire to learn and know about the present and the future, but also to know about our medical forebearers and place them in proper chronological order, realizing the value of their pioneering achievements and thanking God for their perseverence. By studying their works and times, none but the greatest respect develops for them and our profession. Our own rapidly changing field has produced men of wisdom and courage. He who scoffs or decries the doctor of the past for being "old fashioned" shows but his own shortsightedness, bad manners and mental immaturity. Surely, had it not been for our forebearers, how could we possibly be at our present state of knowledge? Only by standing on their strong broad shoulders can our present scientific efforts be justified. They cannot be repaid but we can repay the profession by contributing our best, and making broader and stronger foundations for our successors to stand upon so they can reach greater heights

FORT WORTH HALTER — Dr. W. H. Bickel of Mayo Clinic, guest speaker at the annual meeting of the Texas Orthopaedic Association in Fort Worth April 11, 1960, examines a halter purchased in Fort Worth as members of the association return from a luncheon at the Cattleman's Steak House for scientific session in the afternoon. On the left is Dr. R. A. Murray, Temple, and Dr. Cecil A. Robinson of Kermit is in the center background.

ORTHOPAEDIC OFFICIALS — Dr. David M. Cameron, left, immediate past-president of the Texas Orthopaedic Association, and Dr. Rex J. Howard, Fort Worth, chairman for the Fort Worth meeting, beam enthusiasm at the success of the one day session held in conjunction with the annual meeting of the Texas Medical Association.

of achievements. Then we must act as the "loyal opposition", requiring proof. May none of us have such brittle habits that new truths cannot be accepted.

Respected Repository

"All the world's a stage . . . and one man in his time plays many parts."[2] Today the striving seeker of knowledge; tomorrow the respected repository of recorded facts, who as did Solon, grew "old ever learning many things."[3] Ever a changing panorama—of this is the art composed. "What's past is prologue."[4]

The art is not a passive thing, it is animate, requiring nurturing. With maturation truths are weighed in the scale of knowledge to arrive at a wise solution. Here the key word is **Wise,** this means tact, compassion, retention and recall, interpretation of signs, symptoms and previous experiences, being "—not the first by whom the new are tried—nor yet the last to lay the old aside."[5] Boundless are the joys that come with the **savoir-faire** of the Art. Serenity as learning becomes knowledge; as kindness, consideration and tolerance becomes intimately fused with daily habit patterns. Equanimity with its ever increasing wisdom and truth is passed on to others with loving kindness, fulfilling Christ's teaching as recorded by Luke, —"For unto whomsoever much is given of him shall be much required."[6] I know not how the practice of medicine could become a commercial product whose services could be sold on the open market strictly for gain. Those who try soon loose a commodity to sell. Unless the heart goes with the service the patient does not get full value received. "A good doctor must be a good man."[7]

Prayer of Learned Man

In closing I quote a prayer of a learned man that to me expresses the ultimate in Scientific Art.

"From the irresponsibility that casts aside all old truth in search of nothing better than novelty; from the cowardice that shrinks from all new truth; from the lethargy that rests content with partial truths; and from the arrogance that lays claim to total truth; Oh God of truth, deliver us.—Amen."[8]

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2. Shakespeare, William: As You Like It.
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6. Luke: Bible 12:48
7. Taylor, Rev. E. L. M.: Quoting Herman Nothnagel, Canad. Med. Assn. Journ. 119-122, June 15, 1959.
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Future Doctors Clubs Formed in Albuquerque High Schools

An Albuquerque physician, Dr. E. H. Wood, has organized "Future Doctors Clubs" in several of the Albuquerque high schools. Anyone interested in obtaining information on starting such a club may write Dr. E. H. Wood at 4800 Gibson Blvd., SE, Albuquerque.

New Mexico General Practitioners to Hold Summer Clinic in Ruidoso

The annual summer clinics sponsored by the New Mexico Academy of General Practice will be held in Ruidoso July 18-21. A faculty from the University of Kansas Medical School will present the program. Scientific sessions will be held each morning in Nob Hill Auditorium, with remainder of the day free. The complete scientific program follows:

Monday, July 18
Heart Disease

9:00- 9:30 Clinical Manifestations of Arrhythmias
James E. Crockett, M.D.
9:30-10:00 Heart Disease in Pregnancy
Kermit Krantz, M.D.
10:00-10:30 Difficulties in the Diagnosis of Rheumatic Fever
Franklin C. Behrle, M.D.
10:30-10:50 Coffee
10:50-12:00 Panel Discussion
Drs. Crockett, Krantz, and Behrle
Moderator: Mahlon H. Delp, M.D.
Heart Disease and Air Travel
Anticoagulants
Diets in Heart Disease
Smoking and Heart Disease
Prevention of Rheumatic Fever
Drugs in Hypertension
12:15 Luncheon and business meeting*

Tuesday, July 19
Mother and Child

9:00- 9:30 Jaundice in the Newborn
Franklin C. Behrle, M.D.
9:30-10:00 Forceps and Neonatal Salvage
Kermit Krantz, M.D.
10:00-10:30 Common Congenital Heart Disease
James E. Crockett, M.D.
10:30-11:00 Infectious Hepatitis in Pregnancy
Mahlon H. Delp, M.D.
11:00-11:20 Coffee
11:20-12:00 Panel Discussion
Drs. Crockett, Krantz, and Behrle
Moderator: Dr. Delp
Transplacental Disease
Dangers of the First Trimester
Toxemias of Pregnancy
Immunizations
Poisonings in Children
Cryptic Virus Infections (ECHO)
12:15 Luncheon
Question period to follow

Wednesday, July 20
Cancer

9:00- 9:30 The Physician's Responsibility in the Surgical Treatment of Cancer
Frank F. Allbritten, Jr., M.D.
9:30-10:00 I-131 in the Diagnosis of Carcinoma of the Thyroid
Donald R. Germann, M.D.
10:00-10:30 Chemotherapy of Cancer for the Family Physician
John F. Christianson, M.D.
10:30-10:50 Coffee
10:50-12:00 Panel Discussion
Drs. Allbritten, Germann and Christianson
Moderator: Dr. Delp
Easily Missed Cancer
New Diagnostic Techniques for Cancer
Hyphophysectomy, Adrenalectomy, Oophorectomy
Dangerous Pigmentations
Cigarettes and Cancer
Pain Relief in Cancer
12:15 Luncheon
Question period to follow

Thursday, July 21
Emergencies

9:00- 9:30 Postoperative Medical Emergencies
John F. Christianson, M.D.
9:30-10:00 The Use of X-Ray in the Evaluation of the Emergency Patient
Donald R. Germann, M.D.
10:00-10:30 The Immediate Care of the Injured Patient
Frank F. Allbritten, Jr., M.D.
10:30-11:00 Hepatic Coma
Mahlon H. Delp, M.D.
11:00-11:30 Coffee
11:30-12:30 Panel Discussion
Drs. Allbritten, Germann, and Christianson
Moderator: Dr. Delp
Acute Anuria
Technique of Tracheostomy
Common Causes of Coma
Anaphalactoid Reactions to Medication
Management of Convulsions
Upper Gastrointestinal Bleeding

These presentations are made possible by a grant from the Merck, Sharp & Dohme Postgraduate Program.

Santa Fe Seminar

St. Vincent's Hospital, Santa Fe
February 22, 1960
Harry D. Ellis, M.D., Chairman

Eclampsia and Preeclampsia

Case Summaries: Fred Soldow, M.D.

Seminar Summary: Raymond L. Young, M.D.

On 3/10/55, at 2 a.m., this 20-year-old white woman, primigravida, was brought to the emergency room of St. Vincent's Hospital complaining of swelling of the face and extremities and severe occipital headache. The headache began three days previously. There had been no prenatal care. The expected date of confinement was 4/14/55.

Examination revealed a blood pressure of 160/95, marked eyeground vasospasm, and hyperactive tendon reflexes. The fetal heart rate was 145 B.P.M.

The past medical history was uninforming. There was no known history of prior renal disease.

Shortly after examination, the patient began to convulse. This was relieved by the intravenous administration of one gram of magnesium sulfate.

The patient was then taken to a hospital room and placed on a regimen of complete rest, sedation, periodic intramuscular magnesium sulfate, and ammonium chloride by mouth. During the next eight hours very little urine was obtained but in the succeeding several hours the urine output increased noticeably.

Second Seizure

About 14 hours after admission a second convulsive seizure occurred. This terminated shortly after the intramuscular administration of five grams of magnesium sulfate.

In the next 24 hours, no further convulsions occurred and the urine showed decreasing albuminuria. The patient's general condition was improved and on 3/12/55, labor was induced. A viable two lb. 13 oz. infant was delivered from below. The baby survived. The mother improved steadily following delivery and was discharged in fairly good condition 3/18/55.

Follow-up note: On 3/3/57, the patient was delivered of a second premature infant which survived. There was no evidence of toxemia during or immediately after this pregnancy.

Case No. 2

This 35-year-old, gravida XII, was hospitalized at St. Vincent's Hospital 9/16/59, complaining of epigastric pain. This appeared to follow ingestion of green chile. The pain radiated through to the back. The patient stated that she was eight months pregnant and that she had had no prenatal care.

The past medical history revealed three previous hospitalizations for delivery and during these hospitalizations there was no significant evidence of toxemia. The last delivery was July, 1958, at which time Kahn and VDRL tests were reactive, 64 Kahn units and 16 VDRL dils. being reported. Penicillin therapy was given shortly thereafter.

The patient's temperature on admission was 98.6° and the pulse rate was 60. Following admission the epigastric pain persisted and increased in severity. This was not relieved by antiacid therapy and the administration of Demerol, 100mgms.

A urine specimen was obtained and found to have a dark brown color and analysis of the urine revealed: 1.3 grams hemoglobin/100 ml.; a few RBC/hpf; albumin four plus. The VDRL test was reactive, 2 dils.

Blood Pressure

The blood pressure on admission was 180/130. There was little evidence of peripheral edema, but the eyeground vessels showed marked vasospasm. In addition to demerol, the patient was given Seconal, gr. 1½ and Thorazine, 25 mgm. Later during the day of admission, the patient's epigastric pain subsided and she became drowsy. Her blood pressure remained elevated.

During the night following the day of admission, Magnesium Sulfate was administered intramuscularly at intervals and mild barbiturate sedation was given. The blood pressure remained elevated and the patient remained drowsy.

At about 4 a.m. labor contractions were noted

at 5 to 10 min. intervals, lasting for 30 seconds. At 6:30 a.m. the blood pressure was 160/120 and at 8 a.m. the blood pressure had risen to 190/140. At this time fetal heart tones became faint.

Muscular Stiffness

One thousand ml. of five per cent glucose in water containing 0.5 mgm. of Diuril were started. Magnesium Sulphate (6cc of 50 per cent sol.) was given. The patient's general condition appeared to be deteriorating, and muscular stiffness was observed.

By 9 a.m. the blood pressure had fallen to 140/120. The urine obtained was still dark in color and it gave a four plus reaction for albumin.

About 10 a.m. Apresoline was given. By 11 a.m. the patient was deeply comatose and nuchal rigidity was marked. The blood pressure was now 110/0. Levophed was added to the intravenous of five per cent Glucose solution.

There was no improvement in the patient's condition over the next few hours and she expired at 4:25 p.m. A postmortem Cesarean Section was done and a nonviable fetus was delivered. Permission for postmortem examination was obtained.

Postmortem diagnoses:

1) Massive Intracerebral Hemorrhage.
2) Periportal and Pericentral Necrosis of the Liver
3) Acute Glomerulitis
4) Pulmonary Edema.

Differential Diagnosis: Dr. Margery Whipple:

Eastman's definition of this condition is "An acute disease peculiar to gravid and puerperal women which is characterized by convulsions during which there is a loss of consciousness followed by more or less prolonged coma."

Certainly this is a proper definition and the convulsions are arresting and grave signs. We must, however, consider preeclampsia and eclampsia as the same complex with the convulsions of eclampsia being indicative of progression of the disease. The triad of hypertension, proteinuria and edema occurring in the third trimester of pregnancy or the immediate puerperal period constitute the classical signs of preeclampsia. With progression to convulsions, the status becomes that of eclampsia.

As far as past history is concerned in true preeclampsia or eclampsia there is no renal or hypertensive history. The patients are often young primigravidae but may be any age or parity.

Clinical symptoms noted by these patients are a severe headache of increasing degree, spots before the eyes, double vision, sudden blindness, swelling, especially of the hands and face, rapid weight gain, nausea, vomiting and epigastric pain. The last symptom is often serious in that it may precede a convulsion. This pain in the epigastrum may be central in origin as the pain of tabetic crisis or it may be due to distention of the liver capsule. Langmade has emphasized that it may also be due to esophageal erosion or gastric or duodenal ulcer.

Arterial Hypertension

Physical examination of the patient will show arterial hypertension, by definition above a systolic of 140 mm and/or above a diastolic of 90 mm. Values lower than this should not be ignored if the patient's known normal tension is below the observed values.

Edema, especially of the hands and face, and of the sacral area in a bed patient, is important but perhaps not essential to the diagnosis. Proteinuria is almost always present.

Ophthalmoscopic examination shows changes in the caliber of the retinal vessels with narrowing of the arteries and engorgement of the veins. Additional retinal changes are retinal edema with an increase in light reflex and in some patients flame shaped hemorrhages. Retinal detachment occurs rarely.

The reflexes of the body are usually hyperactive.

In severe eclampsia the patient will often become comatose, demonstrate an elevation in body temperature, pulse and respiration rate, and rather frequently develop pulmonary edema. Oliguria or anuria are common in this stage of the disease.

Proteinuria varies from a trace to a four plus reaction. Erythrocytes may be present from a small number to a sufficient number to cause gross hematuria. Hemoglobin may be present and hyaline and granular casts may be present.

Blood Urea Nitrogen Elevated

The blood urea nitrogen will be elevated in severe cases. The blood uric acid is believed by some to show an earlier rise and the elevation is said to indicate a severe true preeclampsia. Serum albumin and CO_2 combining power may be decreased.

Important in confirming the diagnosis of true preeclampsia or eclampsia is the disappearance within a short time after delivery of hypertension, proteinuria, and edema.

The differential diagnosis is important from a prognostic viewpoint. Essential or renal hypertension (either condition with or without superimposed toxemia) are the main problems in differential diagnosis.

The diagnosis of essential hypertension can be made from the history if its presence is found on examination before 20 weeks gestation. In essential hypertension levels of blood urea, uric acid, and creatinine are infrequently elevated. Essential or renal hypertension occurs more commonly in older multiparae. The presence of renal disease may be known from the previous history.

In recent years the differential diagnosis has been aided considerably by the use of renal biopsy. These were first taken transperitoneally at the time of Cesarean sections and later on a much wider scale by the peri-cutaneous method.

Renal Biopsies

Dieckmann, McCartney, and Harrod at the University of Chicago have reported 145 renal biopsies from pregnant patients. These usually served to differentiate true eclampsia and pre-eclampsia from that superimposed upon pre-existing renal disease. They were able to distinguish five renal conditions exclusive of the typical toxemic lesions of eclampsia and preeclampsia. The follow-up biopsy studies on their patients having true eclampsia revealed a return to normal histologic renal anatomy.

Spargo, McCartney and Winemiller of Chicago have reported cases in which the renal biopsies were studied with the electron microscope. They describe the lesion as "glomerular capillary endotheliosis". They have an eleven-day biopsy which showed some clearing and a return to normal in a follow-up four-weeks old biopsy of the same patient.

Occasionally one is confronted with a case in late pregnancy or immediate puerperium with convulsions and/or coma and no prior knowledge of the case is available.

Dieckman points out, in such a case, that there is nothing characteristic about the convulsions of eclampsia and other conditions such as epilepsy, hypertensive encephalopathy, uremia, hypoglycemia, and meningitis should be considered.

Etiology: Dr. Charlotte Jones:

The etiology of the specific toxemia of pregnancy, namely, the eclamptic syndrome—is unknown. The disease has been studied over the entire world for many years by many investigators, and it is only in recent years that plausible theories have come to light. The present concepts may be roughly grouped into three main theoretical categories: placental ischemia and necrosis, specific toxins, and generalized endocrine imbalance.

The concept of uterine ischemia and necrosis dates back as far as 1914 when it was first considered by James Young. Of late it has occupied the concentrated attention of Jeffcoate, Scott and Browne in London, as well as that of Venner, Bartholomew and others in this country.

These workers feel that increased intrauterine pressure, or possibly some other factor, creates a circulatory change which produces what they term a utero-renal reflex. This reflex acts adversely on the kidney with the result that renal symptoms occur which are typical of the disease entity. Sophian demonstrated this reflex experimentally, and English investigators have evaluated degrees of increased myometrial tonus through the use of radioactive isotopes. From the combined observations it is felt that excessive myometrial tonus through the reduction of the blood supply to the uterus, produces the utero-renal reflex. This reflex, in turn, causes variable vasoconstriction within the kidney and subsequent renal ischemia. As a result splanchnic vaso-constriction occurs producing further impairment of uterine circulation, which inevitably leads to placental ischemia and necrosis.

Placental necrosis is considered pathognomic for this disease. Part of the circulatory aberration in the placenta is centered around the abnormal spasm of the venous sphincters of the placental veins. This, through diminution and obliteration of intervillous spaces results in necrosis of the chorionic epithelium, villus stroma and capillary endothelium. It is felt that the first product of placental necrosis is thromboplastin, but there is suggestive evidence indicating that necrotic areas may well be the source of other unknown endotoxins which enter the maternal blood stream.

Trigger Factor

This brings us to the so-called "trigger" factor exciting the venous spasm. This factor is considered by many to be a direct result of endocrine imbalance. F. J. Browne has written concerning the similarity of the eclamptic state to Cushing's syndrome, and feels that there must be a pressor substance involved that is produced in excess. He has postulated that there is an oxygen sensitive oxidase produced that inactivates the pressor hormones from the adrenal cortex. It is his contension that in the presence of an ischemic placenta one finds lowered oxygen tension and therefore the protective enzyme is made ineffective, and hypertension and other signs and symptoms of toxemia are the result.

Other investigators stress the deliterious results of a change in placental hormones occurring with placental ischemia and necrosis, for example, there is an **increase** instead of decrease in gona-

dotropic gonadotropin and a **decrease** instead of an increase in the secretion of estrogen and progesterone. These workers also maintain that there is excessive hypertrophy of the adrenal cortex in the eclamptic syndrome, and attempt to correlate the resultant increase in corticosteroids and mineral-corticoids with the co-existent sodium retention and hypertension.

I would also like to add that some people have stressed the importance of a psychosomatic background of patients developing preeclampsia and eclampsia. It has also been suggested, but never proven, that the preeclamptic and eclamptic states are correlaries of the Schwartzman phenomena produced in animals.

For as many adherents to the various theories, there are as many opponents, and much more investigation is necessary before we can state clearly the etiology of the eclamptic syndrome, still among the three most common causes of maternal death.

Question: Dr. Howard Seitz:

Dr. Jones, does this problem occur in dogs?

Dr. Jones:

There is a syndrome in pregnant bitches similar to the eclamptic syndrome in the pregnant human, but it appears to last only a few days, and has been demonstrated as being related to hypocalcemia. This is very easily taken care of by the intravenous administration of calcium. The experimental production of eclampsia in animals has been difficult, perhaps because of the difficulty of producing hypertension in pregnant animals. I understand that Assali has produced eclampsia in pregnant sheep.

Pathology: Dr. Harry Ellis:

The pathology of eclampsia is seen mainly in the liver, brain, and kidneys. There are less striking, but perhaps equally important changes, in the uterus and placenta.

The characteristic change in the liver is that of periportal necrosis. Sometimes the necroses extend into the central areas and infrequently the liver necroses become massive. Subcapsular hepatic hemorrhages and intraparenchymal hemorrhages are common.

I should mention that the liver is not a constant site of anatomical change in this condition. Reports vary from a 50 per cent to a 75 per cent incidence. The liver necroses are reported to occur more frequently in patients dying within two days of the onset of the disease as compared to those who die later.

In the kidneys, the common changes occur in the glomeruli and consist of swelling of the glomeruli and partial to complete obliteration of the glomerular capillary spaces. Precipitated protein, often accompanied by red blood cells, is seen in the glomerular spaces. The second case demonstrated the presence of numerous red blood cells not only in the glomerular spaces but in the tubules as well. I would like to show the renal glomerular capillary in eclampsia in a

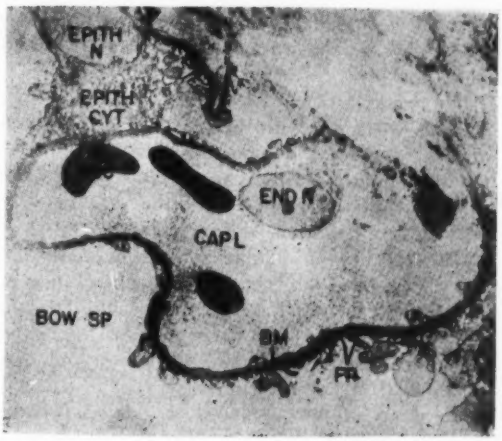

Figure 1. Cross section of a normal glomerular capillary, showing the widely patent capillary lumen and scanty endothelial cell cytoplasm. X 5,000. (Courtesy of Benjamin Spargo, M.D., Chicago.)

Figure 2. A typical ischemic glomerular lobule from a case of preeclampsia. There is marked narrowing of the capillary lumen by the cytoplasm of the endothelial cell. It is clear that the basement membrane is intact and the epithelial change is slight. X 4,500. (Courtesy of Benjamin Spargo, M.D., Chicago.)

section magnified by electron microscopy. Figure 1 shows a normal renal glomerular capillary. The endothelial cell cytoplasm is quite sparse. Figure 2 shows a glomerular capillary loop endothelial cells from a case of eclampsia. Here you see the marked swelling with almost occlusion of the loop lumen by the increased endothelial cell cytoplasm.

Less commonly there develops lower nephron nephrosis. This is said to occur in about one-third of fatal cases of eclampsia. Even more infrequently the kidneys demonstrate bilateral cortical necrosis.

The brain frequently contains numerous petechiae. Gross cerebral hemorrhage occurs and in one series was responsible for death in 47 per cent of fatal cases. The presence or absence of cerebral edema appears to be a matter of some controversy.

The placenta in eclampsia contains more and larger infarcts than normal controls. Zones of necrosis and hemorrhage of the decidua basalis are usually present and there is often associated fibrinoid degeneration and thrombosis of the endometrial spiral arterioles.

There is often an excess of syncytial knots attached to the chorionic villi.

The uterus will usually demonstrate foci of hemorrhage in the myometrium of subserosal connective tissue.

Petechiae are frequently noted in the myocardium and not infrequently elsewhere. Tiny focal areas of myocardial necrosis are reported.

Another pathologic change of interest has been the finding in organs and tissues of the greater circulation, especially the liver, of fibrin thrombi within capillaries and smaller vessels. This has been a variable observation and has not been adequately explained.

Treatment: Dr. Robert Snow:

The attention given to early incipient signs of preeclampsia is probably one of the few really practical developments in the management of toxemia of pregnancy. The best and most effective treatment of eclampsia is prevention by means of adequate prenatal care. Illustrative of the improved results that may be cited are collective studies from New Zealand. There eclamptic deaths have decreased from ninety-three in a period from 1928 to 1933 to one in 1955. Adequate care includes the necessity of regular frequent blood pressure determinations, weighing and examination of the urine for albumin.

Convulsions may occur during the course of a seemingly mild toxemia but are more likely in the severe grades. Most cases occur before or during labor. An eclamptic is an individual whose condition is poor and who is not a good operative risk. The condition demonstrates a tendency to improvement, a tendency particularly to be encouraged by supportive treatment. There is danger from overtreatment especially in too vigorous attempts to rapidly terminate pregnancy. The initial treatment of eclampsia is not concerned with the question of obstetric interference but is a matter of determining the particular form of conservative therapy to be employed.

Absolute rest in bed with a minimum of disturbing procedures is essential. In addition to rest, isolation a darkened room and side rails help to minimize patient stimulation. The blood pressure cuff may be left on. An indwelling catheter is valuable to obtain measurements of urinary output and quantitative determinations of albumin. The following laboratory procedures may be considered: typing and crossmatching, uric acid, npn, serum albumin and CO_2 combining power. These are part of an assessment of the patient's condition.

Danger of Aspiration

Fluids are never given by mouth unless the patient is conscious because of the danger of aspiration. Water and electrolyte disturbances should be corrected. When this is accomplished the fluids intake is usually maintained at 2500-3000 cc depending on the urinary output. Dextrose solutions help to increase the urinary output and supply caloric needs. The salt intake should approximate 1½-2 grams daily. To displace accumulated sodium ion, when the urinary output is adequate and the patient is not in acidosis, ammonium chloride (4 grams three times daily for two days) can be given. Oxygen may be administered during and for 10 minutes after a convulsion.

Magnesium sulfate is the most potent anticonvulsive agent. Parenteral magnesium ion depresses nervous excitability at the neuro-muscular junction in a manner similar to that of curare. It is safe to give 10 grams intramuscularly as an initial dose followed by 5 grams every six hours. Where a more rapid action is desired 1-2 grams may be given intravenously. Precautions are as follows: knee jerks should be tested before each injection. Respirations should be 16 or more per minute. The drug should probably not be continued for more than 24 hours if the urinary output is less than 600 ml. daily. Intravenous calcium gluconate is an immediate antidote.

A good guide to the need for sedation is the presence of hyperactive reflexes. Sedation helps to allay and prevent convulsions. Barbiturates are usually employed and may be given parenterally at first.

The fundamental pathophysiologic phenomena underlying acute toxemia is a generalized arteriolar vasoconstriction with an increase in vascular resistance particularly in the kidneys, liver, brain, and uterus. The use of vasodepressors has not gained uniform adoption but as used by their advocates the aim is to restore the caliber of constricted arterioles to a normal or nearly normal diameter. The route of administration depends on the urgency. Assali recommends Apresoline 30-40 milligrams intravenously to be repeated as often as necessary to keep the blood pressure at 20-25 per cent below control values or as Bryant states at the value existing before or early in pregnancy. The blood pressure is used principally as the means by which therapy is guided. If the blood pressure tends to return toward control values Veratrum or one of the slower acting Rauwolfia preparations may be added to the regimen. Various combinations are employed to obtain the best results and to lessen unpleasant and toxic effects. The veratrum preparations usually recommended are mixtures of protoveratrines A and B. They have less tendency to produce nausea than preparations of the crude drug. The occurrence of cardiac irregularities such as pulsus alternans, gallop rhythm or skipped beats is an indication for stopping the use of veratrum derivatives.

Digitalization is recommended in the presence of cardiac failure or pulmonary edema.

With no convulsions or coma for 48 hours the considerations for delivery correspond to those for a very severe preeclamptic. Delivery when maximum improvement has occurred is preferably solved vaginally with induction of labor by rupture of membranes or with intravenous Pitocin or Syntocinon. Cesarean section is reserved for other obstetric complications or in instances resistant to or unfavorable for induction.

Magnesium Sulfate: Dr. Willis Pickel:

Magnesium Sulfate has been used for many years in the treatment of preeclampsia and eclampsia because of its action as a depressant of the central nervous system. The magnesium ion also causes a dilatation of peripheral vessels with a resultant drop of blood pressure.

The usual adult dosage is 10 cc of a 25 per cent solution given intramuscularly. For a more rapid effect at 6 per cent solution may be given intravenously. The intravenous dosage is 1-2 grams.

Magnesium Sulfate given intravenously has a much shorter duration than when given intramuscularly. Ten gms. of intramuscular Magnesium Sulfate in a 25 per cent solution can be given every two hours with a wide margin of safety. Magnesium Sulfate is effective at a blood level of 4-8 mg/100 cc of plasma where as the lethal dosage is 18-20 mg/100 cc of plasma.

The rate of intravenous injection of Magnesium Sulfate should not exceed 3 cc of a six per cent solution per minute.

Calcium chloride or gluconate for intravenous injection should be on hand to be used as an antidote in the event of respiratory paralysis following the parenteral administration of Magnesium Sulfate.

The patient should be observed carefully for signs of Magnesium overdosage as evidenced by a depression of reflexes, an abrupt fall in blood pressure, flushing of the skin, nausea, or depression of respiration.

Magnesium Sulfate may be used in conjunction with other agents such as Apresoline in the treatment of eclamptic convulsion.

Questions and Comments

Dr. Richard Angle:

Page in his monograph points out that the crux of the situation is to prevent eclampsia. He states that the mortality rate of preeclampsia is almost nil, whereas the mortality rate for eclampsia is in the neighborhood of 10 per cent, sometimes less.

He feels if the disease can be contained to preeclampsia, and its progression to eclampsia prevented the problem has been solved. Since the occurrence of a convulsive seizure is the event that distinguishes eclampsia from preeclampsia, he feels the main effort should be to prevent convulsions.

For this reason, he advocates the use of Dilantin Sodium in cases of preeclampsia and in his experience for the preceding five years there were no cases of eclampsia developing after the introduction of this treatment. Of course, he used other standard treatments, also. But, I think his point is well made. Preeclampsia has a very low mortality, eclampsia has a relatively high mortality and an attempt should be made to prevent the preeclamptic state from progressing. Page further states that in his opinion the preeclamptic state is often undertreated and the eclamptic patient is often overtreated.

Dr. Whipple:

I have not known much about the use of Dilantin in these patients. In reading about eclampsia there is much written about cerebral dysarthria in the convulsing patient. There is said to be a family history of epilepsy or a history

that the patient had convulsions with febrile illnesses in childhood. Many of these patients are said to have abnormal electro-encephalograms.

These background observations would seem to support the idea that Dilantin might have a use in the prevention of convulsions in the preeclamptic. There are preeclamptics that never have convulsions and die, but in general what Dr. Page says is so, once convulsions develop the mortality rate is much greater.

Dr. Bergere Kenney:

In general, in treating patients who have oliguria or in particular acute renal failure, you usually limit rather than push fluids. Are you following a different principle in your treatment of the preeclamptic and eclamptic patient?

Dr. Snow:

Usually after you start treatment the initially low urinary output will increase.

Dr. Ellis:

I think the point of difference in management, here, is that most of these patients do not have or develop acute renal failure.

Dr. Angle:

I want to ask one question about the periportal liver necrosis. Does this sometimes go on to complete necrosis of the liver? Did you say that?

Dr. Ellis:

Yes, I did.

Dr. Angle:

Does this have anything to do with acute yellow atrophy?

Dr. Ellis:

Acute yellow atrophy is an older name for diffuse or massive necrosis of the liver. There are reasons for this replacement of terms. One is that the liver isn't always yellow. The condition called acute yellow atrophy occurs in eclampsia and in many other conditions. I do not know any way to differentiate by pathologic changes, the rare diffuse necrosis occurring in eclampsia from diffuse necrosis occurring in other diseases.

Dr. Angle:

Isn't acute yellow atrophy of pregnancy an example of overwhelming infectious hepatitis occuring during pregnancy?

Dr. Ellis:

I am not familiar with the term "acute yellow atrophy of pregnancy", however, acute yellow atrophy or massive necrosis of the liver as a progression of infectious hepatitis is well known. This progression is more common in pregnancy.

Dr. Angle:

I would like to know if acute yellow atrophy of pregnancy as a manifestation of infectious hepatitis in the pregnant patient is a different condition from the diffuse necrosis that you say is a manifestation of toxemia. I don't know whether they are two separate things or not. What is your opinion?

Dr. Ellis:

Acute yellow atrophy used synonymously as diffuse necrosis of the liver can be and is a complication of toxemia of pregnancy. The problem with patients who are pregnant that contract infectious hepatitis that progresses to diffuse necrosis of the liver is they are more prone to do that than the non-pregnant person, probably for the same reason that they get more severe poliomyelitis. This condition is not the same as the patient who develops diffuse necrosis of the liver in association with eclampsia or preeclampsia.

Seminar Summary: Dr. Raymond Young:

The participants in this seminar have covered the subject admirably, and only a few points remain to be emphasized.

The best way to treat eclampsia is not to let it occur. Once a toxemic patient begins to convulse, the prognosis becomes grave. Not only adequate, but meticulous and painstaking prenatal care, is the obligation of every one of us who attends pregnant patients.

Notice that neither case which Dr. Soldow presented received any prenatal care whatsoever.

While I am not at all sure that the second case was one of the so-called "true" eclamptics, still early and proper prenatal care might well have resulted in a happier outcome.

When the present Chicago Lying-in Hospital was built, Dr. DeLee ordered inscribed in Latin on a plaque in the Doctor's lounge, "Pregnancy in itself places the woman in danger of death". a professor of Classical Languages at the University of Chicago, with a remarkable sense of humor for a Latin teacher, added the initials "G. M., B. L.", meaning "Good Motto, Bad Latin".

Regardless of Dr. DeLee's Latin, his motto is not only good, it should be a fundamental truth to anyone practicing obstetrics.

If one hasn't the time or the inclination to give **excellent** prenatal care, he must not accept the patient in the first place.

Excessive Weight Gain

Excessive weight gain is usually the first warning of true preeclampsia, and it should be treated vigorously. Once true preeclampsia is established, it should be treated **radically.** This means, simply, that if by the 35th week the condition has not responded to conservative measures, then the pregnancy **must** be terminated, either by the induction of labor, or, failing that, by Cesarean section.

The fetuses of toxemic patients do notoriously poorly. They are invariably abnormally small for their intrauterine ages. Statistically, the healthy, even though quite premature, babies of these mothers have a much higher survival rate than the infants who have been exposed for a longer time to the mother's disease **in utero.**

The physician who thinks that "watchful waiting" will result in a large and healthy term baby is simply deluding himself.

Once convulsions have occurred, of course, the pregnancy should be ignored and the disease should be treated conservatively. When the eclampsia has been satisfactorily controlled, delivery by Cesarean section should be performed promptly, but then and only then.

Two Other Points

Two other points remain. First, I feel that the intravenous administration of Magnesium Sulfate should be followed by magnesium blood level determinations. While Magnesium Sulfate is the most valuable single drug we have in treating this condition, its use is not completely without risk.

Second, Dieckman, among others, preached incessantly that **true** preeclampsia and eclampsia were **most** unlikely to recur in subsequent pregnancies, the original toxic episode having occurred in a primigravida. Certainly, sterilization procedures in primiparae with histories of true toxemias and therapeutic abortions in multigravidae with such histories, while still being done in some places, are indefensible. These mothers, rather, should be reassured as to the unlikelihood of recurrences, and should be encouraged to bear more children.

When the etiology of eclampsia is finally defined, I am sure that a multitude of interacting factors will be implicated. Such definition must, of course, also explain the relatively high incidence of eclampsia first occurring, without warning, in the postpartum period.

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ORIGINAL ARTICLES

Porphyrin and Magnesium Supplementation in Collagen and Degenerative Disease

By R. P. WATTERSON, M.D., *Scottsdale, Ariz.*

Specific instructions and medication can be given the patient with arteriosclerotic and atherosclerotic disease that will produce improvement in structure and function. The net effect of these measures must be the improvement of oxidative metabolism and the mobilization and metabolism or excretion of pathologic tissues.[1,2]

Maintenance of an adequate supply of etioporphyrin, Type III, for heme synthesis is essential for continuous replacement of the enzymes of the respiratory chain.[3,4,5,6] Examination of the biochemical patterns in cardiovascular disease indicates interference in synthesis and function of the heme-derived enzyme systems. Attention is especially drawn to the diversion of carbohydrate into cholesterol synthesis when the Krebs Cycle is inhibited, and that fibroplasia is accelerated when oxidative capacity is reduced.[7,8,9] The latter mechanism is recognized in mechanical reduction of blood supply as in Volkmann's Contracture, but is usually not considered when the oxidative block is chemical. Time and space prohibit more complete discussion here of biochemical mechanism. This can be obtained from the references.[10-17]

Fibroplasia and Hypecholesteremia

Since fibroplasia and hypercholesteremia are intimately related to arteriosclerotic and atherosclerotic disease, and since fibroplasia is characteristic of the collagen diseases,[18] the following measures have been correlated into a basic program for patients with the clinical signs and symptoms of cardiovascular disease: — Type III etioporphyrin supplementation to support heme synthesis; magnesium in complexed form to enhance carbohydrate metabolism and inhibit hyaluronidase; reduction of intake of magnesium-low and mineral-low foods (refined sugar, Karo and white flour); reduction of lead intake by elimination of tea, aluminum cookware and tobacco; elimination of milk to withdraw growth-stimulating factors and possibility of viral seeding; substitution of vegetable oils and butter to replace hydrogenated fats, thus increasing the intake of linoleic acid; and maintenance of Vitamin E intake to insure the antioxidant activity provided by the tocopherols.

Long Range Compliance

The necessity for long range compliance with the nutritional selection is explained to these patients. Supplementation with the magnesium and porphyrinic material is specified as necessary of continuation only so long as essential to achieve and maintain structural and functional improvement. The time necessary for this varies with the individual patient, some apparently requiring continuous supplementation. The majority of patients, however, can discontinue medication by the time their weight has reached an optimum level, a consistent exercise schedule has been established, and the functional state of the patient no longer requires support.

The following data was accumulated on twenty-eight (28) cases treated with schedules which included the above-listed measures:

Examination of the data listed reveals consistent normalization of blood pressure without hypotensive medication, improvement in hematocrit and hemoglobin, and consistent reduction of hypercholesteremia.

Clinically the patients evidence increased mental alertness, decrease in musculo-skeletal pain and stiffness, improvement in gross strength and endurance, less shortness of breath on exertion, decrease or disappearance of angina of effort, improvement in skin texture, darkening of the hair, and improvement in peripheral circulation. These changes have been noted in many patients in the 70 to 80 year age group. The majority of these patients reduced or eliminated symptomatic medication.

The above noted clinical and laboratory improvement is more difficult to achieve in the majority of patients without including porphyrin supplementation at least for an initial period of sixty to ninety days. The selection of porphyrin material for clinical use is of major importance. In the author's experience a metal-free, water-soluble potassium salt of etioporphyrin, Type III, from vegetable origin has proven to be the most effective.* The usual dosage was ½ gram daily. The patient response to this therapy indicates that this type porphyrin is utilized in the regeneration of the heme-derixed oxidative enzymes.

Patient	Age	Clinical Diagnosis	Date	Hgb.	Hct.	Choles:	B.P.:	Urine Albu.	C.R.P.
Mrs. D. J.	44	Rheumatoid arthritis	9-17-58	10.5	35	250	120/80	neg.	neg.
		Colitis, chronic	12- 1-58	12.6	40	190	120/80	neg.	neg.
Mrs. H. S.	48	Sinusitis, Psoriasis	1-19-59	12.2	37	242	122/72	neg.	neg.
		Multiple allergies	6- 5-59	13.4	42	176	114/76	neg.	neg.
Mrs. M. B.	72	Osteoarthritis	9- 3-58	11.2	36	216	184/110	neg.	neg.
		Arteriosclerosis	1- 7-59	13	40	180	138/78	neg.	neg.
		Hypertension, Psoriasis							
Mrs. E. M.	53	Rheumatoid arthritis	3- 3-59	10.2	33		118/72	neg.	1+
		Anemia	4- 1-59	13.2	40		120/70	neg.	neg.
Mrs. S. M.	69	Arteriosclerosis	9-17-58	12.2	39	362	220/90	neg.	
		Hypertension	10-15-58	14	42	202	140/82	neg.	
Mrs. B. L.	38	Rheumatoid arthritis	10- 7-58	10.5	36	138	106/60	2+	2+
		Anemia	3- 3-59	13	40		114/72	neg.	neg.
Mrs. M. J.	66	Osteoarthritis	7-22-58	11.6	36	354	126/70	neg.	neg.
		Arteriosclerosis	1-12-59	14	42	196	140/82	neg.	neg.
Mrs. L. G.	69	Osteoarthritis	7-31-58	10.8	37	329	130/80	neg.	neg.
		Arteriosclerosis	10- 1-58	13.5	41	196	130/80	neg.	neg.
		Chronic Sinusitis							
Mrs. M. V.	45	Osteoarthritis	2- 9-59	12.2	39	210	122/74	neg.	2+
			5- 7-59	14	42		114/70	neg.	neg.
Mrs. H. N.	42	Rheumatoid arthritis	7- 3-58	11.9	37	348	104/60	1+	neg.
		Hypothyroidism	1-29-59	14.4	46	196	108/60	neg.	neg.
Mrs. H. D.	65	Rheumatoid arthritis	12- 3-58	11.7	36	198	92/36	neg.	3+
		Hypoadrenia	4-14-59	12.4	39	200	114/68	neg.	2+
Miss S. S.	11	Rheumatoid arthritis	11- 7-58	12.6	40	(Sed. rate 40)		tr.	pos.
			2-12-59	14.3	42	(Sed. rate 26)		neg.	neg.
Miss F. M.	72	Arthritis, mixed	9-30-58	10.7	36	(Sed. rate 22)		neg.	neg.
		Arteriosclerosis	3-24-59	15.4	40	(Sed. rate 18)		neg.	neg.
		Hypoadrenia							
Mrs. M. D.	37	Obesity, Sinusitis	1-19-59	12.6	40	208	154/100	neg.	
		Hypertension	4-3-59	13	40	193	120/80		
Mrs. R. M.	59	Rheumatoid arthritis	1-15-59	10.2	34			neg.	4+
		Hypoadrenia	4- 6-59	14	41			neg.	2+
		Bronchioctasis							
Mrs. C. D.	44	Hypothyroidism	6-16-58	10.5	38	160	84/42	neg.	
		Metabolic insufficiency	3-24-59	14.2	41		116/74	neg.	
Mrs. H. R.	71	Arteriosclerosis	1-23-59	13	40	262	110/70	neg.	
		Osteoarthritis	3-13-59	13	42	180	124/76	neg.	
		Osteoporosis							
Mr. S. A.	5	Fibrosis pancreas	11-28-58	9	30	(Child)			
		Fibrosis pulmonary	3-19-59	13	42				
		Allergies multiple							
Mrs. R. S.	40	Rheumatoid arthritis	11-13-58	10.8	35	117	102/60	1+	4+
			3- 3-59	13	40		104/66	neg.	1+

Patient	Age	Clinical Diagnosis	Date	Hgb.	Hct.	Choles:	B.P.:	Urine Albu.	C.R.P.
Mrs. L. G.	69	Arteriosclerosis	7-31-58	10.8	37	329	124/76	neg.	
		Osteoarthritis	4- 3-59	13.5	41	196	120/70	neg.	
		Chronic sinusitis							
Mrs. C. F.	48	Myxedema, Obesity	11-18-58	11.9	38	254	122/76	neg.	
		Arteriosclerosis	4-15-59	13	40	180	116/68	neg.	
Miss B. Y.	47	Rheumatoid arthritis	1- 8-59	9.6	31		100/70	neg.	4+
		Hypoadrenia	3-11-59	13	40		98/62	neg.	1+
Mrs. M. P.	70	Arteriosclerosis	9-12-58	10.8	38	250	190/80	neg.	
		Hypertension, Myxedema	11-17-58	13.6	41	200	146/80		
		Psoriasis							
Mrs. K. W.	35	Rheumatoid arthritis	11-20-58	11	34		118/70	neg.	
		Hypothyroidism with Myxedema	1-26-59	12.6	39		120/70	neg.	
Mrs. E. C.	76	Arteriosclerosis	11-21-58	9.9	30	320	160/90	neg.	4+
		Hypertensive coronary disease, Chronic anemia	2- 4-59	12.6	40	190	130/84	neg.	2+
Mrs. F. P.	28	Rheumatoid arthritis	7-13-58	11.5	37		100/68	neg.	
			2- 3-59	12.8	40		118/72	neg.	
Mrs. L. R.	60	Rheumatoid arthritis	10-23-58	8.4	30	252	110/72	4+	
		Sinusitis, Hypoadrenia	3-30-59	12.2	39	178	132/70	3+	
Mrs. B. R.	51	Rheumatoid arthritis	9-11-58	10.5	37	210	102/64	neg.	
			4-15-59	13.4	41	180	116/74	neg.	

Withdrawal and replacement of porphyrin and magnesium supplementation and periods of omission and compliance with the other specified measures will convince the patient and the attending physician that these items of the regimen are essential for normalization of intermediary metabolism and maintenance of an optimal functional balance.

Summary

The possibility of devising a total program for correction of basic metabolic faults exists. A program for support of heme synthesis by etioporphyrin, Type III, supplementation, rational dietary selection, restriction of total caloric intake, increased magnesium intake and decrease in lead intake, which has been successfully applied to patients with cardiovascular disease, is presented. Similar long range studies are felt to be the privilege and responsibility of the private practitioners of medicine, since these physicians can best evaluate the long range compliance and response of the patient.

Acknowledgments

*PoChlorin, for etioporphyrin Type III, was supplied by Texophyl Corp., Boling, Texas.
**Hyalex or Cardenz, for magnesium supplementation, was supplied by Miller Pharmacal Co., West Chicago, Illinois.

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THE PRESIDENT'S PAGE

Dr. Deter

"I Am A Doctor"

By Russell L. Deter, M.D., *El Paso*
President, Southwestern Medical Association

"The first indispensible qualification of a physician was that he should have studied well the science of medicine. He should be a man who has read and remembered much of medical subjects; who has great experience of his profession and who hears the case of his patient with calmness; who is painstaking in diagnosing the diseases of his patients; who knows the various bodily organs and understands their function; who treats his patient conscientiously; who is sweet-tongued, gentle, friendly, zealous of the honor of his profession; adverse to protracting to disease for greed or money; who is God-fearing. An ideal healer heals for the sake of healing; he is the best among the healers. The second in rank in the profession is he who practices his art, actuated by the desire for reward and renown in this world. The third, in point of honor, works both for the sake of merit and for money, but gives preference to the first. The fourth in position of nobility in his profession is the physician who rates money higher than merit. The lowest in the skill is the greedy and heartless physician who dishonors his noble profession. The duty of the conscientious physician is to watch carefully the effect of the medicine he prescribed for his patient from day to day; to change and try a still better drug than the one he has already given; to visit the invalid daily, at a fixed hour; to labor zealously to cure him; to combat the disease of his patient as if it were his own enemy."*

Zoroastrian Ideal

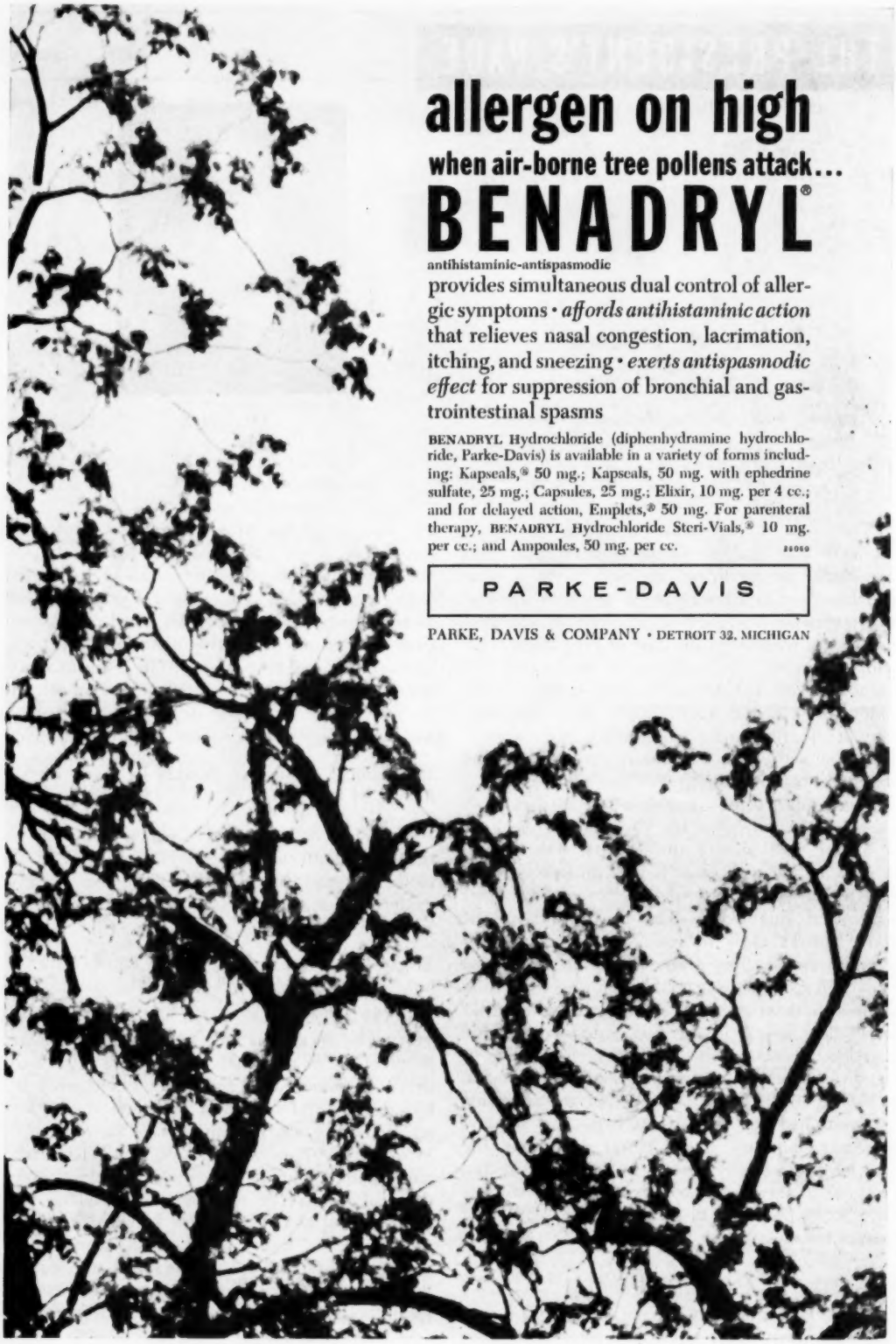
The above statement is the Zoroastrian civilization's ideal for its physicians around the year 600 B.C. in Persia. How little these ideals have changed in some 2,600 years—! As the Bible of the Christian faith and the Torah of the Jewish faith have survived disaster and floods, kings and dictators, revolutions and wars, Nazism, Fascism, Communism and all of the other isms and tricky innovations that have come from puny minds of men in their mad quest for power and an attempt to completely subjugate their fellow men—so has medicine survived these things.

***A Medical History of Persia**—by Cyril Elgood, M.A., M.D., FRCP

Medicine has survived centuries of superstition and faith healing, the mystic era of the medicine man of ancient times and even of modern times, now we face democracy vs. socialism. We shall survive.

Doctors of Medicine

I AM A DOCTOR — so help me, I want my sons to be doctors as long as deep in their hearts they believe in the tenets of medicine as set forth in Persia some 2,600 years ago. Let us stop this business of—"If socialized medicine comes I do not want my son or daughter to be a doctor!" Who do we think we are kidding? What makes anyone think that if medicine is socialized everything else won't be socialized too? As has been said, as long as our thirst for knowledge persists, as long as our desire for research, teaching and the seeking of answers to the cause and cure of disease continues unabated, no one but us will wear the mantle of Aesculapious — we ARE the doctors of medicine.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Clinical Pathological Conference

R. E. Thomason General Hospital, El Paso

Case No. 1364, March 17, 1960

F. P. BORNSTEIN, M.D., *Editor*
Presentation of case by VICTOR BLANCO, M.D.

History: Dr. Nathan Kleban:

A 50-year-old unemployed cook was admitted to the hospital on October 29, 1959. Six weeks before admission the patient began to experience increasing weakness and absence of appetite. He denied abdominal pain, vomiting, diarrhea or other symptoms.

The patient had been in good health until the present illness.

His father died at age 34 of tuberculosis. His mother died of heart disease at age 65. A grandfather had died of cancer of the stomach at age 65. One sister had died of scarlet fever and another of diptheria.

Physical Examination:

T. 100; P. 96; R. 24; B. P. 104/64.

The patient appeared chronically ill. The right pupil was larger than the left, but both reacted to light. A firm, non-tender, movable mass was felt in the epigastric region. The left hypodhondrium was tender and resistant to palpation.

Hospital Course:

Daily maximum temperatures were about 102. Parenteral fluids were administered. Although the patient moaned and stated he was uncomfortable, he denied having pain. He refused oral fluid and food, was restless but unable to turn himself. On November 12 an operation was performed.

Laboratory Findings:

Blood counts: 10-29-59—WBC 11,100, Hb. 16.3 gms., Ht. 48 vol.%, Stabs. 2, Segs. 72, Lymphs. 21, Monos. 4; 11-5-59—Hb. 15.5 gms., Ht. 46 vol.%, WBC 12,200, Eosino .2, Stabs. 2, Segs. 71, Lymphs. 24, Monos. 1; 11-11-59—Hb. 12 gms., WBC 7,400, Eosino. 4, Stabs. 1, Segs. 67, Lymphs. 26.

Urinalyses: 10-30-59 — Yellow, cloudy, acid S.G. 1.014, Albumin 1+, Sugar negative, WBC 5-10/hpf, many sq. ep. cells, numerous bacteria—bile negative; 10-11-59—Yellow, cloudy, acid, S.G. 1,005, Albumin 1+, sugar negative, WBC 20-25/hpf, RBC abundant, many sq. ep. cells, many bacteria, few urates; 10-31-59—Urine culture—negative.

Chemistry. 10-31-59—Amylase—68; Glucose—77 mg.%; Van den Bergh Direct—.235, Indirect —.435. 11-1-59—Calcium—3.7 mEq/L; CO_2 capacity—15 mEq/L; Chlorides—94 mEq/L; Potassium—3.7 mEq/L; Sodium—122 mEq/L. 11-2-59—CO_2 capacity—23 mEq/L; Chlorides—82 mEq/L; Potassium—4.3 mEq/L. 11-12-59—Chlorides—16 mEq/L; Potassium—3.2 mEq/L; Sodium—132 mEq/L.

Febrile agglutinations: 11-2-59—Typhoid O & H—negative; Paratyphoid A & B—negative; Brucella and Proteus OX19- negative.

Thymol turbidity: 11-3-59—1.0 unit.

Blood Culture: 11-5-59—negative.

Feces: 10-29-59—Trace occult blood, amoebiasis negative; 11-2-59—negative for parasites or ova.

Serology: 10-30-59—Negative.

Ceph. Floc.: 24 hours negative, 48 hours 1+.

X-rays: 11-2-59—Chest, barium enema—Conclusions: Displacement of the large bowel by an extrinsic mass. Retention of dye in the left kidney with obstructive change suggest a retroperitoneal origin of the mass.

11-5-59—IVP—Findings as described are consistent with a mass lesion in the left kidney and should be further evaluated by retrograde examination on this day. No definite function is demonstrated on the right, however, a very heavy gut shadow causes considerable interference with the examination. Since no dye is seen in the bladder shadow, the examination indicates poor renal function.

X-Ray Discussion: Dr. Vincent Ravel:

The urogram was done September 30 and was unsatisfactory technically. A second examination, a barium enema, was done the second of November and the dye that was instilled over 48 hours before was observed. Now the barium enema

itself is not remarkable except for the displacement of the colon by a very large retroperitoneal mass which apparently is connected to or indistinguishable from the kidney. At this time we were able to see the grossly hydronephrotic findings of the calyceal system.

Another intravenous urogram confirmed the previously hydronephrotic left kidney. At no time were we able to visualize the calyceal and pelvic system of the right kidney, or the ureter on the right. This may be of course obscured by the heavy colonic soft tissues and the gas in the colon.

There are also some other secondary findings such as calcification in the iliac vessels secondary to a peripheral arteriosclerotic vascular process. You can all see the appendix well on the barium enema. Fundamentally we have a large retroperitoneal soft tissue mass on the left, indistinguishable from the lower pole of the left kidney.

This probably represents a carcinoma, although it could be a cyst of the kidney. The other soft tissue masses of this size which might be considered are pancreatic cyst or enlargement of the spleen, but this is unlikely.

Clinical Discussion: Dr. Victor Blanco:

After I saw the X-rays it changed my way of thinking. The only thing that I knew for sure from the protocol was the fact that the patient's profession didn't have anything to do with his illness. The history is very meager. The patient had increasing weakness and absence of appetite, no abdominal pain, vomiting or diarrhea or other symptoms. The physical examination states that the right pupil is larger than the left and both react to light. To me that does not mean anything. The patient had a firm, non-tender, mobile or movable mass felt in the epigastric region, if it was in the epigastric region?

In the X-rays to me that is not epigastric region. I worked up the protocol as assuming that there was a mass in the epigastric region, but from the X-rays I can see that this mass covers the entire middle abdomen. Actually it is mostly below the epigastric region. The mass is not described except that it is mobile and non-tender, it doesn't say what type it is, whether it is smooth or nodular.

X-Ray Findings

So I am going to have to go on from the X-ray findings, because what I considered previously couldn't possibly be what this man has got, except a retroperitoneal mass.

As to the laboratory work, this man is not anemic, his white count is not elevated out of proportion, the urine shows a 1+ albumin and a few white cells per high power field, and many bacteria, bile was negative. Evidently the man was worked up to see if he had any liver metastasis as a Van den Bergh was ordered. His amylase was normal.

His Van den Bergh is .235 direct and indirect is .435. Well, the direct, as I understand it, should be negative and the indirect should be between .25 and 1, so that lies within normal limits. The calcium lies within normal limits. The first reading of the CO_2 combining power is 15 mEq/L which is extremely low and that signifies that the man had acidosis. His chlorides are 94, potassium 3.7, and his sodium 122 mEq/L.

Chemical Disturbances

Now evidently these chemical disturbances were corrected to a certain extent by giving him IV fluids and it is possible that his electrolytes are slightly below normal because the man had not been eating. How much he had been eating I do not know. The protocol states that the man has not had any vomiting or any diarrhea.

The febrile agglutinations are negative. Thymol turbidity is normal, blood culture is negative, the feces examination is negative for occult blood, the sedimentation rate is normal. The cephalin flocculation is 1+ within 48 hours which does not mean too much as far as parenchymal damage is concerned. There was no BUN done. X-rays studies were started on the patient right away.

A chest was done, and a barium enema. When I read the protocol I was surprised that an upper GI was not done since the mass was supposed to be in the epigastrium. On the 5th of November they die the IVP which showed no definite function on the right side and the left showed retention of dye in the kidney. The patient must have had some type of obstruction of the ureter specially on the left side and as you can well see the mass lies mostly to the left side.

Retroperitoneal Mass

Therefore I will have to take this as a retroperitoneal mass. I had thought it might be a pancreatic cyst but I have never seen a pancreatic cyst this large and the patients usually have some abdominal pain, specially after eating, and vomiting. This man had neither. His only other complaint as stated in the protocol is abdominal discomfort and the patient did not say that he had any pain.

The differential diagnosis of retroperitoneal mass involved a great number of possibilities. In

the first place, it could be metastatic. There is no mention of a genital examination, the patient might have a tumor of the testicle, with extensive metastases retroperitoneally. I think that this mass is benign because it is freely movable.

Now I believe that the tenderness and guard which he had in the left hypochondrium is probably from the kidney, representing retention or obstructive symptoms of the kidney. Now what are the differential diagnoses of benign retroperitoneal masses. As I said before, I am going to have to go just from what I have seen.

The most common condition that I have seen is lipoma. This is a soft mass, it appears cystic, it is mobile. Although these masses are usually locally malignant, if excised totally the patient will get rid of the tumor.

Multilocular Cysts

It could be a cyst. I have seen kidneys with multilocular cysts; papillary cystadenocarcinoma of the kidney gives you a mass this large, and the mass is mobile simply because it is a locular mass filled with fluid. Fibrosarcomas or any of the nerve tissue tumors are usually fixed. I considered a neuroblastoma because of the pupils being unequal, but these tumors are usually found in children. I believe this mass is entirely too large for a pancreatic cyst. I even thought about aneurysms, until I saw the X-ray pictures.

So my best bet is that it is a benign retroperitoneal tumor, what it is, I don't know.

Dr. H. M. Gibson:

Dr. Blanco said all the essential things that can be said. Is the film of the first IVP available at all? Was there such a film? Can we see kidney shadows on that at all? The question here is what is the situation in that right kidney in addition to the trouble on the left side. What I am looking for is a film where we might get a reasonable idea whether there is a right renal shadow present. There is one diagnosis that I might add to those of Dr. Blanco and that is a crossed renal ectopia. So we have to assume if the urea nitrogen was this elevated that there must be some damage to the right kidney, or the right kidney is lacking. If we are to believe the laboratory work we have to assume that this is not a malignant process and that it is not a very badly infected process. I refer specially to the sedimentation rate. The sed. rate is notoriously sensitive in any renal condition, infection, or tumor, for that matter. So I am going to point out the possibility that this could be a congenital defect with a crossed renal ectopia, with the right kidney attached to the lower pole of the left kidney. Can anyone top that?

Dr. Solomon Heller:

I don't intend to top it. I think the most likely possibility is a huge pancreatic cyst. If the hemoglobins have any accuracy they drop rather strikingly from 16.3 to 12 grams on 11-11-59, therefore this mass may be the result of rather massive bleeding into a pancreatic cyst. The renal changes may represent a secondary pressure phenomenon.

Dr. W. R. Gaddis:

Unfortunately I am prejudiced because I have seen this patient. Dr. Akan and I presented this man on what was then Saturday morning rounds. I palpated this mass and if it will clarify the situation any for you, Vic, in my opinion it was nodular and it was very hard and the extent of motion as described here is very insignificant. I would say this also in respect to the laboratory procedures, they must have gotten the wrong blood when they took the sedimentation rate because I didn't believe it then and I don't believe it now.

Dr. Blanco:

All I want to say is how misleading such a protocol can be. Dr. Heller mentioned the fact that the man's hemoglobin was 16 grams and a couple of days later it was 12, well, the man probably had a hemoconcentration, he was given I-V fluids and his hemoconcentration dropped.

Dr. Licon:

I just wanted to ask Dr. Ravel how serious he was when he remarked that this might be a gumma on account of the opaque material around the buttocks.

Dr. Ravel:

That wasn't mentioned on the X-ray report but there were several films that showed opaque media in the buttocks which frequently represents bismuth injections for lues, and I just facetiously remarked, "Maybe it's a gumma."

Dr. Saul Appel:

For those in the back of the room who didn't hear it, there was a BUN done and it was 28, which of course is slightly elevated. We do not know on what date it was done. I agree with Dr. Blanco that the man was probably dehydrated on admission and yet it is very interesting that the admission urine was 1.014 specific gravity and 12 days later 1.05. On the latter time we can assume that he was well hydrated.

The electrolyte changes are rather interesting, too, if you will notice the low CO_2 capacity, the low chlorides, potassium and sodium shortly after

admission. As time went on the sodium rose a little bit and the potassium appears to get up to 4.3 but on the 12th of November it was down to 3.2 mEq/L which is low. An EKG would have been interesting to help clarify the potassium level and I might say that an EKG is a lot cheaper, quicker to obtain and sometimes more reliable in many laboratories, than the serum potassium. I am not trying to make a diagnosis at this stage of the game but I think that in a patient like this where you know that the potassium is low on one day, it is a good idea to follow it very carefully and to obtain EKGs and thereby you will have some control level from which you can make rough estimates from day to day. You don't have to get all 12 leads, if you just get the routine leads one, two and three every moning, it gives you a pretty fair idea of what his potassium level is in the myocardium and that is a pretty fair reflection until you can get the finer tests done later in the day.

Dr. Jack Postlewaite:

Well, I think this would be an amazing interpretive problem if you are going to do electrocardiograms to balance this man's electrolytes. In the first place, his potassium is low. Second place, his calcium is low, which has a very definite change on the cardiogram. In the third place, he has been chronically ill with malnutrition. Then you will notice the sodium is low, you will notice he is in acidosis and also in early uremia.

Dr. Appel:

The changes in uremia that occur in the EKG are so typical and so simple to recognize that given an unknown electrocardiogram, one can put a question down, is the patient in uremia? The effects of potassium on the T-waves are very easily seen. Calcium merely prolongs or shortens the Q-T interval. Therefore in a patient with the usual findings in uremia, namely, a low calcium and a high potassium, you get a very prolonged Q-T interval, following the R-way and then a peaked T-wave, it's really quite pretty, and in this patient I think it would be helpful anyway.

Dr. Bornstein:

Dr. Gupta, will you please report the surgical findings?

Dr. R. P. Gupta:

We made a left paramedian upper quadrant incision and a great big tumor was found as soon as we entered the peritoneal cavity. The omentum was involved and the stomach and part of the liver was involved in it and we thought that it was arising from the kidney and a biopsy was taken and then we closed.

Clinical Diagnosis: Abdominal mass.

Dr. Blanco's Diagnosis: Benign retroperitoneal tumor.

Pathological Diagnosis: Retroperitoneal reticulum cell sarcoma.

Pathological Discussion: Dr. F. P. Bornstein:

The first slide, Fig. 1, shows an extremely cellular and rather undifferentiated tumor. This is from the peritoneal biopsy. You can see the anaplastic, malignant, more or less uniform round cells, so a tentative diagnosis of a tumor in the lymphosarcoma group was made. Fluid and cell blocks made from the fluid received also revealed obvious malignant cells. The patient died one week later. There were no significant findings in the chest.

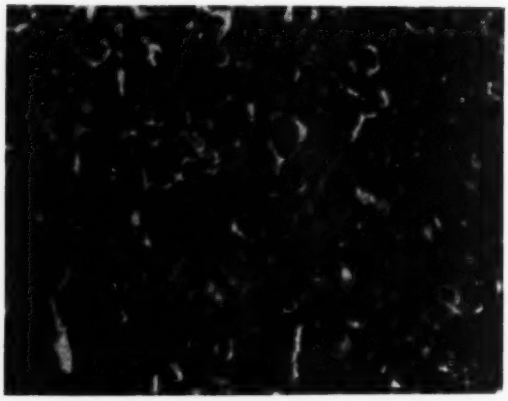


Figure 1

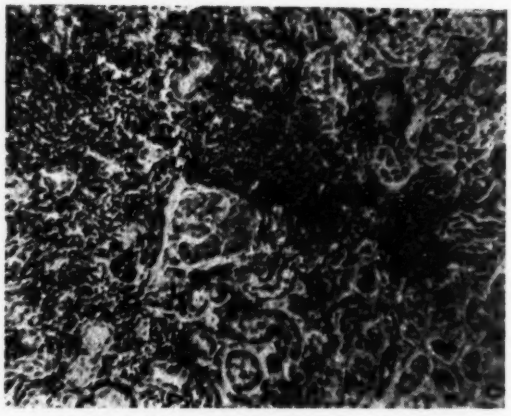


Figure 2

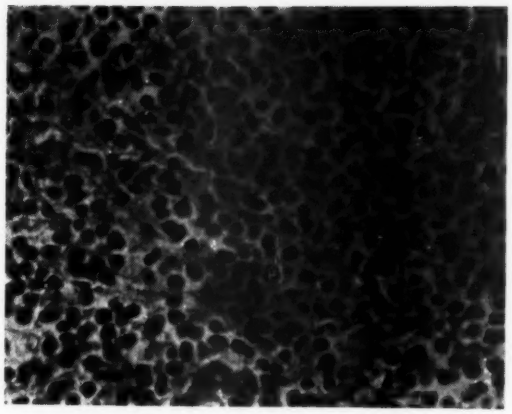


Figure 3

There were extensive metastatic lesions in the peritoneum, including the visceral peritoneum. However, the intestinal mucosa was intact. Obviously, therefore, this was not a primary tumor of the gastro-intestinal tract. A large retroperitoneal tumor was found measuring about 20 x 25 x 15 cm. This tumor involved the entire vena cava and left kidney.

Faint Outline

On sectioning only a faint outline of the kidney was demonstrable, with a minimal amount of renal tissue. It was therefore impossible to determine whether this was a primary renal tumor on the left side or whether this tumor arose retroperitoneally and involved this kidney secondarily. The right kidney was also involved and here sharply circumscribed, distinct metastases were easily visible.

The spleen weighed 600 grams. The follicles were greatly enlarged and at one pole was a hemorrhagic nodule measuring 4 cm. which represented metastatic tumor. Histologically the tumor was obviously malignant, obviously invasive and sufficiently differentiated to be classified as a reticulum cell sarcoma. (Figures 2 and 3)

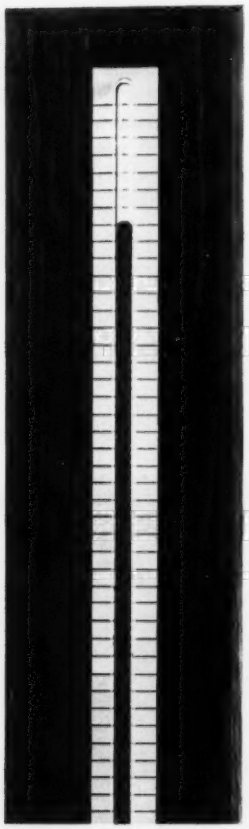
In summary, we are dealing with a patient who rather quickly developed a rather enormous tumor mass which arose from somewhere in the peritoneal space. This tumor destroyed one kidney rather completely and metastasized to the other kidney, the spleen and the entire peritoneum.

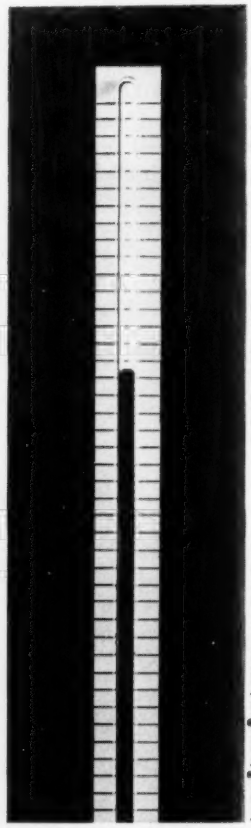
MEDICAL OFFICERS — Shown at the recent meeting of the Texas Medical Association in Fort Worth are, left to right, Dr. Milford Rouse, Dallas, past president of TMA; Dr. Louis M. Orr of Orlando, Florida, president of the American Medical Association; Dr. May Owen, Fort Worth, newly-elected president of the Texas Medical Association; and Dr. Franklin W. Yeager of Corpus Christi, immediate past president of TMA. Not shown is Dr. Russell L. Deter of El Paso, elected vice president of Texas Medical Association.

for a smooth downward curve

New Rautrax-N results in prompt lowering of blood pressure.[1] Rautrax-N, a new and carefully developed antihypertensive-diuretic preparation, provides improved therapeutic action[1] plus enhanced diuretic safety for all degrees of essential hypertension. A combination of Raudixin and Naturetin, Rautrax-N facilitates the management of hypertension when rauwolfia alone proves inadequate, or when prolonged treatment, with or without associated edema, is indicated.

Naturetin, the diuretic of choice, also possesses marked antihypertensive properties, thus complementing the known antihypertensive action of Raudixin. In this way a lower dose of each component in Rautrax-N controls hypertension effectively with few side effects and greater margin of safety.[1-16]





Other advantages are a balanced electrolyte pattern[1-16] and the maintenance of a favorable urinary sodium-potassium excretion ratio.[2-16] Clinical studies[1-5] have shown that the diuretic component of Rautrax-N—Naturetin—has only a slight effect on serum potassium. The supplemental potassium chloride provides additional protection against potassium depletion which may occur during long term therapy.

Rautrax-N may be used alone or in conjunction with other antihypertensive drugs, such as ganglionic blocking agents, veratrum or hydralazine, when such regimens are needed in the occasionally difficult patient.

Supply: Rautrax-N—capsule-shaped tablets providing 50 mg. Raudixin (Squibb Rauwolfia Serpentina Whole Root) and 4 mg. Naturetin (Squibb Benzydroflumethiazide), with 400 mg. potassium chloride.

Dosage: Initially-1 to 4 tablets daily after meals. Maintenance-1 or 2 tablets daily after meals; maintenance dosage may range from 1 to 4 tablets daily. For complete instructions and precautions see package insert. Literature available on request.

References: 1. Reports to the Squibb Institute, 1960. 2. David, N. A.; Porter, G. A., and Gray, R. H.: Monographs on Therapy 5:60 (Feb.) 1960. 3. Stenberg, E. S., Jr.; Benedetti, A., and Forsham, P. H.: Op. cit. 5:46 (Feb.) 1960. 4. Fuchs, M.; Moyer, J. H., and Newman, B. E.: Op. cit. 5:55 (Feb.) 1960. 5. Marriott, H. J. L., and Schamroth, L.: Op. cit. 5:14 (Feb.) 1960. 6. Ira, G. H., Jr.; Shaw, D. M., and Bogdonoff, M. D.: North Carolina M. J. 21:19 (Jan.) 1960. 7. Cohen, B. M.: M. Times, to be published. 8. Breneman, G. M. and Keyes, J. W.: Henry Ford Hosp. M. Bull. 7:281 (Dec.) 1959. 9. Forsham, P. H.: Squibb Clin. Res. Notes 2:5 (Dec.) 1959. 10. Larson, E.: Op. cit. 2:10 (Dec.) 1959. 11. Kirkendall, W. M.: Op. cit. 2:11 (Dec.) 1959. 12. Yu, P. N.: Op. cit. 2:12 (Dec.) 1959. 13. Weiss, S.; Weiss, J., and Weiss, B.: Op. cit. 2:13 (Dec.) 1959. 14. Moser, M.: Op. cit. 2:13 (Dec.) 1959. 15. Kahn, A., and Grenblatt, I. J.: Op. cit. 2:15 (Dec.) 1959. 16. Grollman, A.: Monographs on Therapy 5:1 (Feb.) 1960.

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New Concept of Retirement Inaugurated in Brownsville Home

A new concept of retirement created for individuals with a record of public service in the field of medicine has been inaugurated at the Luearlam Manor in Brownsville, Texas.

The Manor is a non-profit retirement home located in the semitropics on the border of Old Mexico, near the Gulf. It differs from most retirement homes in that it seeks to provide a haven in later years primarily for those individuals whose life has been marked by a high degree of public service to their trade or profession, but who have reached retirement age with insufficient savings to maintain the standard of living enjoyed throughout their working career.

Luearlam Manor was built from an idea conceived by the late Earl Corder Sams, president and chairman of the board of the J. C. Penney Company. Funds which he left the Sams Foundation in Brownsville provided a number of public benefits, among them the Luearlam Manor. The Manor is neither a charity home nor a nursing home. On the nonprofit basis, accommodations range from $135 per month to $185 per month for private quarters and fine food in the luxuriously furnished and gracefully landscaped single-story building. Revenues go back into operations.

Complete Facilities

Facilities include complete installations for hobbies, crafts and the arts, private dining rooms, community hall and library, individual snackbars and kitchens, and in general, the accoutrements of gracious living with as much participation in the community life of the Manor as desired by the individual.

The person eligible for the Manor is not "typed." The hope is for a self-contained group of creative people still active in retirement and functioning as a dynamic civic force in the community or area. The Manor is administered by the Sisters of Mercy, who also operate the adjoining Mercy Hospital in Brownsville. The Manor itself is non-sectarian and open to all who otherwise qualify. It is not a religious retreat.

Financial arrangements made with guests are also somewhat different from most homes. Entry is on a mutually acceptable basis after a three month trial. No property entailment, financial pledges or endowments are asked, and the individual retains complete freedom of action. The home is open to single men and women and married couples, with special private apartments to cover all circumstances.

The home has been open for some time on a trial basis, but active invitation to interested parties was begun only this year. Those interested in investigating the home can do so by writing directly to Luearlam Manor, Box B, Brownsville, Texas. All inquiries are personally answered and all correspondence is completely confidential.

Dr. Jenness of Texas Western Publishes Volume of Poetry

Franklin B. Jenness, M.D., director emeritus of the student health service of Texas Western College of El Paso, is the author of a new book of poems entitled "Spindrift and Sagebrush" soon to be released by the Naylor Co. of San Antonio.

Dr. Jenness had previously published three volumes of poetry, "Man O' War Rhymes," "Sea Lanes," and "Ocean Haunts." Many of his poems have been printed in anthologies and in publications throughout the nation.

SOUTHWESTERN MEDICINE has published several scientific papers of which Dr. Jenness was the author.

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